venture offensive brown pus was found. Then, through an oblique incision, the terminal half of the last rib was removed and the diaphragm cut horizontally; it was thickened and inflamed. Forceps were passed upwards and forwards along the needle into the abscess cavity, which was the size of an orange, and lying chiefly behind the right lobe of the liver. It was drained through a large tube, and the patient made a good and rapid recovery.

CASE III

T. B., a male aged 59, had had indigestion and attacks of vomiting for years. On November 1st, 1922, he was seized with sudden severe abdominal pain, accompanied by vomiting, and was admitted to Guy's Hospital on November 4th. The right upper quadrant of the abdomen was rigid, and there were signs of compression of the base of the right lung. The x-ray report stated: "Right diaphragm is greatly elevated and completely immobile. Left diaphragm moves well. Gas bubble above right lobe of liver." This abscess was explored transpleurally by resecting the tenth rib in the mid-axillary line, four and a half pints of fluid being removed. The patient died on December 11th, and at the post-mortem examination a large right empyema and collapsed lung were found. The subphrenic abscess was secondary to a perforated duodenal ulcer.

CASE IV

I. G., aged 33. A gastric ulcer was operated upon seven hours after perforation, suture of ulcer being done with suprapubic drainage. Eight days later pain was complained of below the left costal margin, and examination proved a "rub" at the left base. A straw-coloured fluid was aspirated. On the eleventh day a left subphrenic abscess was drained by incision below the last rib. On the fourteenth day there was rib resection for left-side empyema; the patient died on the twenty-eighth day.

CASE V

C. McC., aged 62 years, was admitted to Guy's Hospital on May 2nd, 1925, with a fifteen-hour history of acute pain in the upper abdomen. Laparotomy revealed a perforated duodenal ulcer. Suture with drainage of the peritoneum was performed. Three weeks later he had pyrexia with pain over the lower ribs on the right side. The white blood count was 20,000. X-ray report: "Right leaf of diaphragm is slightly elevated, movements greatly diminished. Slightly indefinite opacity at right base suggestive of pleural thickening, but no effusion seen. Left base and left diaphragm show no abnormality." Pus having been found by an exploring needle on June 11th a portion of the eleventh rib was resected on the right side; the lower margin of the pleura was found and sutured to the diaphragm. The diaphragm was then opened subpleurally and the abscess drained. He made a satisfactory recovery, and was discharged a fortnight later.

CASE VI

K. S., aged 38, was admitted to Guy's Hospital on September 12th, 1927, with fourteen days' history of pain in the left loin, with pyrexia. There was tenderness in the left loin and dullness at the left base of the lung, but x rays showed no abnormality. An exploring needle inserted through the tenth space located pus apparently below the diaphragm. A portion of the eleventh rib was resected and the pleural cavity opened; no pus was present. The pleura was closed and the diaphragm incised and a subdiaphragmatic abscess drained. One month later the pyrexia was still present, and pus was found by an exploring needle in the left pleural cavity; this was drained through the original wound. After a prolonged convalescence the patient left the hospital with a discharging sinus.

CONCLUSIONS

A review of the causes, symptoms, and diagnosis of the subphrenic abscess is given.

The treatment is discussed, and the posterior subpleural method of approach is advocated in order to avoid pulmonary complications.

Cases are cited illustrating the complications following the transpleural route. Cases II and V show the advantages of the subpleural route.

REFERENCE

¹ H. L. Barnard: *British Medical Journal*, 1908, i, 371, 429.

GENERAL OBSERVATIONS ON SEA-SICKNESS AND THE LABYRINTHINE THEORY*

BY

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The British nation, eminently seafaring, is conspicuous for its neglect of the study of sea-sickness. In consequence of this ignorance there is as much diversity of opinion about cause as there is about treatment. The very prevalent view that sea-sickness is a matter of mere nerves arises from the observations that vomiting sometimes occurs before the passenger has embarked, and also that sea-sickness is apparently so often controlled by suggestion. There are other views equally superficial which overlook the primary cause because of some casual feature which is unduly prominent. So it is with treatment. The layman, in the person of the old traveller or the bedroom steward, is always prepared with an "infallible remedy." Even the medical man, at sea as on shore, may have partial success with a favourite drug, and, turning a blind eye to failures, may become stereotyped in his advice. The underlying factors are altogether too inconstant for certitude. Personal adaptability, the subsidence of winds and waves—any one or all may make a remedy appear effective. Thus it is advisable that the would-be voyager, when urged to try so many "personally recommended cures," should bear in mind the poverty that lies in such wealth.

CONDITIONS AT SEA

It is exceptional to have seas continuing rough for a trip of six days. It is not uncommon to hear sailors speak of a wind as "blowing itself out"; rough days alternate with calm days; all the greater care is needed, therefore, in waxing enthusiastic over a specific remedy. The weather may have changed, the sea subsided, the first attack of vomiting have corrected the overtaxed digestion, or one's sea legs may have come to the rescue. For these reasons it is necessary to appraise all the accessory aids as nearly as possible in order to judge how far any particular remedy has contributed towards recovery. Care must also be exercised as to the denotation of sea-sickness in order to be quite sure that we are considering *sea-sickness* and not *sickness at sea*—cases of mere "nerves," those who are "sea-sick" when they step on the gangway or even (as in one case) when in sight of the sea, those who vomit merely in imitation or in consequence of the odour of vomiting, all in fact, belonging to the readily suggestible, but who do not properly come within the scope of the inquiry.

THE SHIP'S MOVEMENT

Quite a large number of old travellers (ship captains among them) never acquire complete adaptability to ship movement. One ship surgeon of fourteen years' standing never put out to sea without, for the first two days, headache, mental confusion, and nausea, which went on to vomiting if the sea became rough. Long familiarity with one type of ship, in susceptible persons, may give undue confidence in their capacity as good sailors, only to have this rudely shattered as in the case of so many "old salts" when transferred during the war from big battleships and big liners to destroyers and trawlers. One may venture to say that with the exception of certain rare and mostly pathological cases, all normally constituted people are susceptible to some definite type of movement. Nevertheless sea-sickness has not the terrors for passengers it had in old times. Our parents may have been hardier, but the descriptions of crossings

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in the days of the sailing ships and early steamers make nauseating telling. The present-day liners, with their size, their exquisite balance, the diversion by exhaust of all unpleasant odours from engine and from galley, and the immense precautions with regard to cleanliness and comfort, make of the ordinary crossing a genuine rest and holiday. It is not possible for a ship to be so constructed as to keep an even keel at sea. A ship's stability, its reputation as a good sea boat, depends to a certain degree on its harmonic response to wind and wave. The tendency has lately leaned towards giant liners, as they are unaffected by the wave movement which smaller craft find definitely uncomfortable, although these big vessels, with a certain type of sea, may also roll and pitch to an unpleasant degree.

CAUSATION OF SEA-SICKNESS

An inquiry into the causes of sea-sickness was held with Group-Captain Martin Flack, Dr. J. R. Marrack, and Major Reid. The ship chosen has a splendid reputation as a sea boat, but fortunately for our investigations the outward journey was the roughest crossing experienced in it for over eight years. Major Reid was enabled, with a ship-movement indicator and his rate-of-turn gyro, to analyse into its components the movement of the ship in a head sea, twice registering a maximum pitch of 10 degrees; with a sea abeam, a maximum roll of 20 degrees was recorded. The yaw—a sort of "side-slip"—was also taken. In bad weather the rolling and yawing are interrelated. The observations not charted were the scend, or upward and total displacement of the ship, and the oscillation. Needless to say, the scending throughout the trip was always present, the others—pitch, roll, and yaw—varying in combination with it. The most unpleasant movement is what is called "corkscrew," when the seas are on the bow or quarter, a combination of roll and pitch on a basis of yaw and scend.

Major Reid's apparatus was able to register the time-frequency of the roll, pitch, and yaw, which will later be seen to have considerable significance when comparing ship movement with rotation experiments. Any statement of rolls exceeding 30 degrees should be received with caution. But even a big roll, if slow in development and change of direction, need not necessarily be unpleasant; the type that provokes sea-sickness is generally the suddenly unexpected arrest of movement and change of direction. Whatever the ship's movements may be, the passenger can only partially counteract it by bodily adjustment, since the movement takes place in so many planes. Lying "fore and aft" for roll or "athwart" for pitch is well advised, but no fixed position does more than give a relative measure of ease. Since the change of direction cannot be anticipated, the eye fails to pick up datum levels which will rectify the erratic information from the semicircular canals. In very rough weather, the ship's movement may not be provocative at all; the boat may not pitch, roll, or scend, for a wind of sufficient force may flatten out the waves. It is generally in the aftermath of the gale that the sea begins to rise.

The sea voyager, then, is submitted to movements from without which he bears ill, contending with others than those in the horizontal plane to which he is well accustomed in car and train. The experiences that have something in common with ship movement are those of a quick-moving lift, of the aeroplane in an air pocket, of the swings and mountain railways of the world's Coney Islands, and, perhaps most of all, of a camel, which rolls and pitches. All these have movements in other planes than the horizontal, but with a sensible duration in each; after a pause they abruptly pass to other planes. This is obviously entirely different from, say, the quick jerk up and down of the parade cavalry trot, which, although the abdomen is rudely shaken, has never been known to

cause symptoms similar to sea-sickness. All movements such as these are altogether too short in duration to be provocative.

A number of subjects, eight in all, were examined for pulse rate, ocular imbalance, and blood pressure, before and after they were seated for fifteen minutes on the automatic trotting horse. They were all new to this experience, and were told not to rise to the movement, but relax in order to get the maximum of jolt from a rise and fall of the seat from 5 to 6 inches. In no case, although the abdomen was thus submitted to a fairly severe tax, was there change in any of the readings; the pulse rate was normal, and there were no symptoms of nausea, vertigo, or even headache.

The point to note with regard to the movements of the ship and of allied means of transport is that there is an appreciably long latent period before the development of symptoms. The threshold is lowered by a summation of stimuli, the symptoms even under bad conditions usually occurring after ten minutes, and often not before three or four hours have elapsed. This sharply distinguishes the genuine sea-sick type, whose distress is definitely due to the ship's movement, from those highly suggestible persons who retch the moment they set foot on the gangway, or are affected by the sound, the sight, or the odour of vomit.

SYMPTOMS

The symptoms differ very considerably from case to case. A list of these was drawn up and found to include for the most part blurred vision, a feeling of visual uncertainty, giddiness, coldness of the extremities, headache (variable in position), cold sweats, hot sweats, mental confusion, lethargy to any effort, mental or bodily, salivation, nausea and vomiting, occurring in any order within certain limits. The cases were roughly classified as severe, moderate, and slight; because vomiting is regarded as a classic symptom of sea-sickness it was made the standard for severe cases; those cases with one or two not very distressing symptoms, such as vertigo, lassitude, etc., were put down as slight; headache, nausea, and any other symptoms which were definitely uncomfortable were placed in the moderate group. The examinations were made in conjunction with Dr. Martin Flack, the result being: severe 86, moderate 11, slight 27.

Symptoms with their Frequencies March, 1930

Vomiting	86	Hot and cold sweats	
Vomiting without		in turn	5
nausea	34	"Feeling of heat" ...	1
Vomiting with nausea	52	"Feeling of cold" ...	1
Nausea	74	"Sinking feeling" ...	2
Nausea without		Lassitude	6
vomiting	22	Light-headed	3
Headache	63	Constipation	2
Vertigo	47	"Acid stomach" ...	1
Mental confusion ...	32	Thirsty	1
Pallor	40	Pain in back	1
Blurred vision	17	Diplopia	1
"Hot sweats"	25	Asthma	1
"Cold sweats"	5		

A Compilation made by One Surgeon over the Period 1924-26

Vomiting	320	Vomiting without	
Nausea alone	240	nausea	60
Headache	165	Lassitude	44
Vertigo	122	Blurred vision ...	40
Abdomen discomfort...	97		

PHYSIOLOGICAL TESTS

With regard to the simple physiological tests that were practicable, three symptoms were appropriate. The first was blurred and confused vision. Dr. Flack deals with this aspect, examining the claim that ocular disturbance is a primary factor in sea-sickness. It is well known that certain patients (keeping the eyes on a datum level, such as the horizon) derive considerable benefit by closure of the eyes, by bandaging one eye, by wearing coloured glasses, or even by paralyzing accommodation with a local application of atropine. That these remedies fail in the great majority of cases is now admitted, but that the eye may be a contributory factor

is not to be denied; the question, however, is to what extent. The Bishop Harman apparatus was therefore used to test imbalance before and after sea-sickness, and before and after experiments in rotation with the Bárány chair. There was little doubt that a large majority of early cases were associated with an initial heterophoria, and that this definitely increased with the development of sea-sickness. Nevertheless, the monoculars did not escape sea-sickness, neither did the zero Bishop Harman cases, so that perfect eye balance is not necessarily a protection, nor is single vision.

44 sea-sick cases showed B. H. readings above 3.

66 sea-sick cases showed B. H. readings below 2.

4 non-sea-sick cases showed B. H. readings above 3.

24 non-sea-sick cases showed B. H. readings below 2.

That the eye can be involved without the cerebral effort of visual attention is demonstrated by rotating the subject in the Bárány chair with closed eyes. Nystagmus is very apparent under the closed lids, but the confusion and vertigo are considerably increased when the eyes are opened, owing to the visual information still further conflicting with the other proprioceptors and extroceptors. In sea-sickness the closed eyes undergo a constant and compensatory adjustment of equilibration, despite the absence of any object in the visual field. This adjustment acts, not only in correspondence with the waves of pressure in the endolymph of the labyrinth, but to a lesser degree with the tonic neck reflexes and the tactual sensations from the parts involved in posture. Such bodily adjustments of eye and musculature have all been described by Magnus,¹ under the headings of "tonic neck reflexes," "tonic labyrinthine reflexes," and "optical righting reflexes."

The next investigation was that of the pulse rate and blood pressure. The signs and symptoms themselves indicated the need of such observations. Some subjects showed extreme pallor and slight collapse, others a heightened colour, sweating, and a dilated pupil, suggesting either a parasympathetic or a sympathetic disturbance. Sometimes these alternated, parasympathetic symptom following sympathetic. In others it would seem as though the cases displayed certain features of each. It was often difficult to decide on the type of disturbance. In the better-defined cases the pulse rate showed either a bradycardia, with a drop in rate when the subject stood up, or a tachycardia still further increased on standing. The blood pressure showed a corresponding coincidental change, some cases demonstrating a blood pressure with a systolic reading below 100, and others with a systolic pressure above 140 when the normal was 120 or thereabouts, all being young and healthy people. These are shown in the following tables.

Alteration of Blood Pressure after Certain Prominent Symptoms

Symptoms	Blood Pressure		No Change
	Fall	Rise	
Severe	20	16	8
Moderate	6	—	—
Slight	2	3	3

Alteration of Blood Pressure, with Severity of Symptoms

Vomiting (severe)	21	14	9
Nausea	17	11	24
Headache	20	12	7
Giddiness	10	9	6
Confusion	10	4	4
Cold extremities	—	—	—
Hot sweats	9	8	3
Other symptoms	16	16	14

Last of the observations were the biochemical tests. Three years ago, after some interesting statements on sea-sickness by Dr. Oriel in the *Lancet*, there followed in the *British Medical Journal* an extraordinary number of conflicting opinions. Dr. Oriel's results seemed in sharp conflict with expert opinion. They were interesting and unorthodox, but perplexing in that a biochemical irritant, a condition of ketosis, was suggested as the possible cause of sea-sickness. The question to investigate, then, was whether ketosis was invariably present as a cause of the symptom of sea-sickness, or whether it was an effect of the vomiting or of starvation. Dr. Marrack, on two separate Atlantic trips, undertook to make some examinations on cases of sea-sickness prior to vomiting, after vomiting, and also on some non-sea-sick controls. The report of this investigation is made the subject of a separate paper by Dr. Marrack (p. 178).

OTHER EXPLANATIONS OF CAUSE

Many views of the cause of sea-sickness, though aired in the medical press, hardly deserve consideration, since they are conjectures only, and are supported by neither clinical observation nor experimental evidence. Such are the views of the primary importance of cerebro-spinal ebb and flow, cerebral congestion and anaemia, the varying pressure changes in hepatic circulation, the long omentum; these possess only a speculative interest. Then there is the opinion that is based on the success of treatment. Respiratory drill is the result of the view that the causal agency in sea-sickness is respiratory arrhythmia. In the first place this arrhythmia is not very marked except in highly agitated individuals, and is at variance with the fact that on shore, except in very abnormal subjects, no amount of distress, terror, or any emotion, brings about symptoms such as vertigo, nausea, or vomiting. In the second place, directing attention away from the dreaded object to some task or effort requiring attention occasionally succeeds. In the third place, even making every concession, the drill is of service to the very few, usually only those of apprehensive type; for that reason it has fallen into disrepute. Lastly, like so many remedies, it depends too largely on personal suggestibility to be generally valuable.

Dr. Poulton, in his interesting observations on the causes of nausea, in the Oliver-Sharpey Lectures for 1925, on certain visceral sensations, considers the locality of nausea to be in the back and lower part of the throat, and that it is due to a condition of tension. He describes the sensation as one of sinking in the throat, and views the condition as caused by a tug or distortive action of the oesophagus on its fixed portion at the rigid framework, the skull and vertebral column. The sensation of sinking he ascribes to a rise of tonus of the stomach wall. He produces experimental evidence to show that the stomach may actually fall, coincident with the sensation, and that bracing up the abdomen may wholly check it. Dr. Poulton considers that the nausea of sea-sickness is attributable to the movement of the ship, particularly by its retardation during the latter part of the descent. He is not prepared to say how far the acceleration at the beginning of the ascent may also account for it. This is a very reasonable presentation of the possibility of a peripheral factor in the production of nausea. That it may be a contributory element one is prepared to admit, but the hypothesis does not explain why nausea may be extreme in the very sea-sick patients who keep to their bunks or who wear a tight belt. With regard to the locality of nausea it is, as Dr. Poulton states, most difficult to get definiteness of opinion. Many describe it as vaguely extending over the upper part of the chest and throat; that the pharynx is here involved is probably due to its extroverted association with the pharyngeal reflex of vomiting.

THE LABYRINTHINE THEORY

The normal reactions of the vestibule are responsible for righting reflexes which compensate active displacements of the body. They are excited not so much by abnormal postures of the body as by movement, its initiation, its cessation, its acceleration, and its retardation. The loss of the associated reaction (the righting reflex) in the airman when flying, all unawares, upside down in a cloud, is evidence that the saccule and utricle of the labyrinth have lost their one-time delicate appreciation of static relations, so that the function of the labyrinth in man is now almost entirely confined to the semicircular canals.² The righting reflexes evoked by vestibular excitation are the same as those involved in the general static reactions; they are the extraocular compensatory adjustments, and the statokinetic reflexes from the neck and body as a whole. The musculokinetic system differs from the labyrinth in that it is aesthetic, and subject to inhibition, whereas the labyrinth has no associated labyrinthine sensation, and is entirely outside voluntary control. The musculokinetic reflexes therefore resemble a great many other reflexes, such as the conjunctival and the pharyngeal, which can be inhibited. The rider, when thrown from his horse, must inhibit these reflexes, which may destroy him, and the diver must deliberately counteract these influences the moment he takes off for a head dive. The statokinetic reflexes indirectly excited by the vestibule are then subject to voluntary control, which may explain certain facts in connexion with immunity from the after-effects of excessive and repeated stimulation.

It does not seem to be clearly understood how limited is the range of effective vestibular response. A long ancestry of adaptation to a stable environment has not fitted the vestibule with a defensive mechanism against some types of excitation which cause complete disorientation. From the very nature of lymphokinesis it will be realized why continuous rotation may fail to register, or at the cessation of rotation why movement of the endolymph, still continuing, may transmit information which is in conflict with that of the other exteroceptors and proprioceptors. When the labyrinth is destroyed, as it is in some deaf mutes, there is no longer a vestibular impulse, and in consequence neither conflict nor disorientation. The inherent failure of the labyrinth to register certain kinds of excitation bears witness to the body's limited ancestral experience of movements other than those self-initiated. Waltzing is no exception to this, inasmuch as, though at first self-initiated, it afterwards depends on the momentum which ensues, and which thus places it in the type of movement imposed from without.

There is considerable confusion, then, as to the proper function of the labyrinth. Nystagmus and postural and kinetic deviation are frequently regarded as if they were normal responses, whereas they are an expression of dysfunction and complete disorientation. It is nevertheless true that these, and not the normal responses, are material for the clinician, for paradoxically it is only by the abnormal response that the normality of the vestibule can be determined.

The tests employed to determine the integrity of the vestibule are those of caloric douching, of galvanic stimulation of the ear, and of body (= head), rotation; they are all abnormal or unusual excitations, and in the normal labyrinth bring about disorientation. The vestibule alone of all the organs engaged in the function of equilibration is abnormally susceptible to this kind of excitation, and the above reactions follow immediately on cessation in the case of rotation, and in a few seconds after the initiation of caloric douching and galvanic stimulation; they are called the primary reactions, and in the case of rotation usually

disappear in less than thirty seconds. Apart from these primary and abnormal reactions there are secondary signs and symptoms depending upon the severity of the experiment and the susceptibility of the subject, and they continue after the primary reactions have passed away, the symptoms sometimes lasting for twenty-four hours. The secondary signs are a rise or fall of blood pressure and of pulse rate, and heterophoria; these signs rarely persist for more than thirty minutes. The secondary symptoms are variable, but vertigo, nausea, and headache are common. These secondary signs and symptoms are unquestionably evidence of irradiation, which is due either indirectly to the immediate results of excessive stimulation from the conflict brought about by nystagmus, postural and kinetic deviation with other exteroceptor and proprioceptor impulses, or directly to the excessive vestibular impulses in and by themselves. They are in no sense true reflexes, despite the ability in animal experiments to obtain invariably such vasomotor, cardiac, and respiratory effects, along with intestinal peristalsis, nausea, and vomiting.³ They are no more specific than are other very frequent examples of irradiation.

THE SEMICIRCULAR CANALS AND RELATIVE
SUSCEPTIBILITY

Both experience and experiment indicate the remarkable difference in the secondary effects after stimulation of the vertical, as compared with the horizontal, canals. To adequate and normal stimulation the verticals respond as effectively as do the horizontals, but for excessive stimulation there is a noticeable difference. It is unusual for waltzing, merry-go-round movement, and all such horizontal rotation to cause these secondary signs and symptoms, except in very rare cases, whereas scenic railways, swings, lifts, a rough sea, affect unpleasantly a large number of people. In experiments with the Bárány chair, in which the subject is seated with the head in different planes, according to the canals chosen for excitation, it is found that the abnormal excitation of twelve rotations in the horizontal for most people causes little discomfort, though the primary responses are more pronounced than when the vertical canals are tested. Twelve rotations, however, of the latter cause intense discomfort, the sense of disorder is overwhelming, and nausea and vomiting are quite common.

In attempting roughly to group subjects according to their susceptibility to abnormal excitation, it is remarkable how few display secondary effects from horizontal rotation. Only two of the many subjects showed a response; one vomited, and the other (the ship surgeon previously mentioned) felt malaise for twenty-four hours afterwards. Such persons so affected are usually subject to train- and car-sickness, and dislike waltzing. Then there is a large number who even enjoy these movements, but are upset by the swings, scenic railways, fast-moving lifts, and passenger ships in a moderately rough sea. The next group are not upset by the foregoing, but succumb to a torpedo-boat destroyer or a trawler in a gale, and to excessive vertical canal stimulation by rotation or caloric douching. Lastly, there are the pathological cases, the deaf mutes with functionless labyrinths, who show neither primary nor secondary effects in response to any kind of excitation.

NYSTAGMUS AND VERTIGO

The only normal and functionally useful reflex between the vestibule and the eye through the medium of the nuclei of the third, fourth, and sixth nerves is the eye-righting reflex. Owing, however, to the objective evidence of nystagmus arising from abnormal excitation of the vestibule, this connexion between the eye and the vestibule has been called the nystagmic circuit. The disorientation

associated with this abnormal excitation has been considered to be due either to a conflict from the disorderly exteroceptor, the visual impulses, or to the disorderly proprioceptors—the extraocular impulses—rather than to the disorderly vestibular impulses, in and through themselves. That it is not entirely dependent upon the former or visual impulses is clear from the fact that vertigo may still occur on shutting out the visual field by closure of the eyes; that it is not entirely dependent on the extraocular impulses is evident from two observations, the first being that after the disappearance of the primary reactions—that is, nystagmus, postural and kinetic deviation—vertigo may still persist; the second being that vertigo may follow abnormal excitation of the vestibule in the absence altogether of the primary reactions. Malan⁴ of the Italian Air Force has observed that nystagmus and the primary reactions after abnormal excitation of the vestibule by rotation may, after a time, fail to appear on frequent repetition of the rotation experiment. This observation is in accord with my own experience, but, unlike Malan, I do not consider this is due to fatigue, but believe that it depends on cerebral control, an increasing ability to inhibit the effects of the abnormal excitation of the vestibule.

It is in most cases easy to curtail the time limit of nystagmus by asking the subject of the experiment to focus his vision persistently on some near object, and the same result observed by Malan in his subjects will, in due course, occur by a deliberate act of concentration rather than by an effort of self-preservation and eye fixation acquired, little by little, to eliminate the unpleasant effects of nystagmus. This same feature is associated with nearly all superficial and some organic reflexes, which, as stated previously, may come under cerebral control. The opinion has been advanced that the vestibule can have no relation to sea-sickness because nystagmus has not been observed in sea-sickness, while it is inevitable in chair rotation. This is erroneous, and is an entirely inadequate statement, and the basis on which the comparison is made has no norm. Rotation, when conducted so as to simulate ship movement, can produce the syndrome of sea-sickness without any of the primary reactions. Two errors in the argument depend on failure to collect evidence as to the time periods of the various ship movements, such as the pitch or the roll, and on ignorance of the importance of choosing for abnormal excitation the same canals that ship movement excites—that is, the vertical canals. With regard to the time element, this can be approached from the point of view of the time of ship movement, as compared with the time taken for the usual experiment with horizontal rotation. The most vertiginous of simple ship movements is the pitch, which in one phase rarely exceeds five seconds, only a third of the time taken by eight continuous horizontal rotations, which occupy sixteen seconds. To simulate the pitch, three rotations only should have been made, but then nystagmus would not have appeared in most subjects. In the next place, the pitch throws excitation on the vertical canals so that the comparison should have been instituted between ship movement and excitation of the vertical canals rather than with the horizontal. Had this been done another remarkable fact would have been forthcoming, and that is that eight rotations in the verticals produce a period of rotatory nystagmus of six seconds, or vertical nystagmus of eight seconds, according to the position of the head, against fifteen seconds of horizontal nystagmus. It can now be understood why three continuous rotations only of the verticals are unlikely to elicit the primary reactions such as nystagmus. Neither the individual phases of a pitch or roll on the one hand, nor a corresponding period of vertical canal rotation on

the other, will therefore cause primary reactions, but it is true of all these excitations that if they are repeated again and again the secondary reactions will almost unfailingly occur. Thus it is that the experiment of rotation with the head 120 degrees forward or 60 degrees backward for three continuous revolutions, including a pause of two seconds with the head brought to the erect in the interval, continued for two to four minutes, will cause these secondary reactions without the primary ones.

Of the eight out of the fifty-two subjects who consented to this variant to the usual experiment of the eight continuous rotations, all but two were nauseated, with mental confusion and headache, and they showed the physiological reactions in blood pressure and pulse rate changes, together with imbalance. There is therefore the closest resemblance between ship movement and rotation experiments when done in this way. In both there is a latent period, and both depend upon a summation of stimuli causing the secondary reactions—the syndrome, in fact, of sea-sickness.

The results of caloric douching dispose of the possibility of any visceral or somatic origin of sea-sickness. The same train of symptoms occurs as after rotation, but with caloric douching the head and body remain fixed throughout the experiment. That does not preclude the possibility of the visceral element, as in ships or lifts, acting as an accessory factor and aggravating a disorder already in being from bulbar irradiation. There are, in fact, no exteroceptor or proprioceptor impulses which may not directly or indirectly assist in the development of secondary signs and symptoms, but there are few cases, and exceptional ones, where such impulses act in and through themselves; for the most part, they play the part of signalizers. It is for the above reasons that overemphasis of the causality of the nervous and apprehensive factors of auto-intoxication by dyspepsia and constipation appears disingenuous. These, with ocular susceptibility, the toxæmia of tobacco, alcohol, and of the menopause, derangement of the cerebral circulation by mitral disease, and very many other conditions, may render the subject abnormally susceptible to this type of movement, or may aggravate the disorder once set up.

TREATMENT OF SEA-SICKNESS

For the therapy of sea-sickness it is therefore obvious, as with every other type of dysfunction, that not one of these factors can be omitted from consideration. From this it must appear superfluous to state that suitable measures against these may, and often do, avert an attack. But the matter does not rest always thus simply. In the failure so to avert sea-sickness, having taken all reasonable anticipatory measures, we have still the problem of its treatment when established. There is, of course, the treatment by direct and indirect methods of suggestion, with a certain amount of success to its credit, but with failure also, as is self-proclaimed in the ever-increasing number of advertised remedies. That these latter meet with both success and failure is due to a lack of discrimination, and it is imperative that a surgeon should not label his case "sea-sickness" and proceed to prescribe the last and most fashionable remedy. If he does so he will meet with some success, but also with inevitable disappointment, which will display his ignorance of the varying and conflicting manifestations of the disorder.

An examination of the secondary reactions is therefore obligatory, but it requires effort and a study of the case. That these reactions present anomalies has already been apparent in the analysis of sea-sick cases, but is still more obvious in that of rotation cases. The statement for specific therapy will not be complete without a brief summary of these experiments.

ANALYSIS OF SECONDARY REACTIONS

In order to analyse the secondary reactions it was decided to record the results from eight continuous rotations on fifty-two volunteers, as offering a wider field for research, only eight having consented to the variant of two-minute interrupted rotations. The subjects were all healthy. All but twelve were between the ages of 17 and 30. Of the twelve, the ages ranged between 40 and 52, ten of these with a long sea service, and two with air service. The primary reactions, being irrelevant, were not recorded.

The results were those usually found in sea-sickness, the blood pressure taken for convenience as the chief vasomotor reaction, and the symptoms and signs were tabulated alongside as follows:

Systolic Blood Pressure	Symptoms and Signs	Nil
Rise	13	13
Fall	8	12
Unchanged	2	6

It will be observed that just half of the subjects showed a rise, indicating that the experiment was not too severe. Twenty showed a fall, and eight remained unchanged. Some of the twenty-six subjects were possibly apprehensive, and thirteen of these showed symptoms. Another curious feature is that while twenty showed a drop in blood pressure, twelve of these apparently showed no signs or symptoms. Of those whose blood pressure remained unchanged, eight in number, six were long-service Navy men who remembered being sick only as young lads. One of the eight was an airman who, for later and more drastic experiments, remained abnormally insensitive, though he showed the usual primary vestibular reactions. Three long-service Navy men showed a fall in blood pressure, without symptoms, so they said; these admitted susceptibility in heavy weather.

NOTE ON SEA-SICKNESS*

BY

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Experience has shown that there is little connexion between a history of sea-sickness and the possibility of being air-sick, except in persons who are invariably severely sea-sick. Candidates for aviation, therefore, are not asked whether they are subject to sea-sickness, but whether they are subject to severe sea-sickness, as well as unduly sensitive to rotary or uneven movement, such as those on roundabouts, switchbacks, or scenic railways, etc. They are also questioned as to train-sickness and tram-sickness.

To determine suitability for aerobatics, the following test has been devised. The subject is seated in a rotating chair, and, while sitting as in an aeroplane, with eyes open and head in the best possible position—that is, looking inside the knee further from the direction of spin—is subjected to a relatively fast spin, ten times in twenty seconds. The pulse rate and arterial pressures are taken just before the spin, and immediately afterwards. In special cases, the effect upon the ocular muscle balance is also investigated. The main results of the test, which was worked out in the Royal Air Force Medical Research Laboratory in 1922–24, may be summarized as follows:

1. In fit pilots, particularly those accomplished in aerobatics, rotation produces but little effect upon the

An examination of the sea-sick cases reveals the fact that the number of cases with a fall in blood pressure exceed those with a rise—twenty-eight with a fall, and nineteen with a rise. In the rotation experiments, on the other hand, there were thirteen with a rise and eight with a fall. There is a column headed Nil (no symptoms) showing that rotation influenced blood pressure without causing symptoms in twenty-five cases. The analysis of sea-sick cases shows no parallel column, but it is reasonable to assume that a great many voyagers might exhibit the same blood vascular reaction without necessarily exhibiting symptoms, and would therefore not come under notice in the ordinary way. It should also be noted that in eleven out of the fifty-eight sea-sick cases no change of blood pressure was exhibited.

CONCLUSION

This presentation of the subject of sea-sickness is not intended to be destructive of all existing remedies. It is rather directed against the assumption that any drug or method of treatment is infallible, and is incidentally intended to impress on the ship surgeon the need for careful appraisal of type, and, when prophylaxis fails, analysis and evaluation of signs and symptoms. Only after this can he avoid drugs and methods which are definitely contraindicated. Intervention is sometimes unwise, and sea-sickness may occasionally be a blessing; on the other hand, relief can be confidently offered to urgent cases from among the many valuable remedies which may be clearly indicated.

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pulse rate or arterial pressures, and does not disturb the eye muscle balance.

2. In cases subject to vertigo, nausea, or vomiting, rotation produces a marked rise in pulse rate and systolic and diastolic pressures; the eye muscle balance is also greatly disturbed in such cases.

3. In subjects liable to fainting, rotation produces marked pallor, and a characteristic fall in the diastolic pressure.

4. In highly strung subjects, liable to develop an anxiety neurosis, there is a marked anticipatory rise of pulse rate and systolic blood pressure prior to rotation, which may or may not be further affected by rotation.

5. In practice, it is found that subjects may tend to exhibit a combination of abnormal responses, but the main criterion is the fact that the satisfactory pilot is relatively stable in all his responses.

A relevant case as regards sea-sickness is given here. The subject was a naval officer who had suffered from sea-sickness in submarines and torpedo-boat destroyers.

Case showing Liability to Vertigo, etc.

The subject was 24 years old, and the official report stated: "On each occasion that I have taken this officer in the air for the purpose of dual instruction, after fifteen minutes he has collapsed through sickness." In an examination on June 20th, 1923, before being placed on the rotating chair his pulse varied from 6–8 in five-second periods, his systolic blood pressure being 128 mm. Hg and his diastolic 82 mm. Hg. After rotation, his pulse, in five-second periods, was 11, 12, 11, 12, 11, 12, and did not fall back below 10 in one minute; the systolic blood pressure was 172 mm. Hg, and the diastolic 98 mm. Hg. The nystagmus time was normal, and the ocular muscle balance was greatly disturbed.

* Read in the Section of Medicine at the Annual Meeting of the British Medical Association, Winnipeg, 1930.